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14. ABSTRACT With support from the Air Force Office of Scientific Research, we made significant theoretical and empirical advances in our understanding of cognitive control. We discovered new phenomena and developed theories to account for them. We developed theories of cognitive control and visual attention that integrated mathematical psychology with cognitive science and with neuroscience. We published our work in major journals, including Psychological Review, Journal of Neuroscience, and Journal of Experimental Psychology. We trained several graduate students and postdoctoral fellows and strengthened our collaboration with Tom Palmeri.						
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Final Report: Air Force Office of Scientific Research Grant FA9550-07-1-0192,  
“Modeling the role of priming in executive control: Cognitive and neural constraints.”

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With support from the Air Force Office of Scientific Research, we made significant theoretical and empirical advances in our understanding of cognitive control. We discovered new phenomena and developed theories to account for them. We developed theories of cognitive control and visual attention that integrated mathematical psychology with cognitive science and with neuroscience. We published our work in major journals, including *Psychological Review*, *Journal of Neuroscience*, and *Journal of Experimental Psychology*. We trained several graduate students and postdoctoral fellows and strengthened our collaboration with Tom Palmeri.

The aims of our research were to advance our understanding of the computational and neural mechanisms that underlie cognitive control. The research focused primarily on the stop-signal paradigm, in which subjects engaged in a primary “go” task are asked to inhibit their response when a stop signal is presented. This task has been studied extensively in cognitive science, neuroscience, and clinical science in a variety of species including humans, monkeys, and rodents. The grant supported publication of two reviews of the literature on the stop-signal task, one on theory (Verbruggen & Logan, 2009a) and one on data (Verbruggen & Logan, 2008a), and a publication describing a program for running the stop-signal task that we made freely available on the internet (Verbruggen, Logan & Stevens, 2008). We conducted empirical and theoretical research on the stop-signal task and we extended our modeling effort to visual search.

The empirical work focused on control adjustments subjects make following trials on which stop signals are presented. Subjects typically slow down on trials following stop signals (Bisett & Logan, 2011; Verbruggen et al., 2008). Our research documented the conditions that lead to slowing and distinguished among theories of the factors that produce the slowing. We found evidence of post-error slowing, in which subjects slow down when they fail to inhibit but not when they inhibit successfully (Verbruggen et al., 2008). We also found evidence that subjects slow down when they succeed at inhibiting (Verbruggen et al., 2008; Verbruggen & Logan, 2008b, 2008d). We found that repetition of the stimulus in the go task was an important factor determining post-stop-signal slowing. We found post-error slowing on trials on which there were no stimulus repetitions and post-success slowing on trials on which there was stimulus repetition. The role of stimulus repetition suggested the hypothesis that subjects formed associations between stimuli in the go task and their goals, associating a go stimulus with stopping if it occurred on a stop-signal trial. When the stimulus repeated, the association was retrieved and produced a tendency to inhibit the response, which slowed go response time (Verbruggen et al., 2008). We showed that these associations can persist for up to 20 trials following the initial pairing of the stop and go stimulus (Verbruggen & Logan, 2008d), suggesting that long-term memory is involved in control adjustments. We extended our goal-association hypothesis to the “go/no-go” task, in which subjects are

presented with one of two categories of stimuli and have to respond when one category is presented but not when the other is presented. Many researchers assume that the go/no-go task involves response inhibition, like the stop-signal task, so we hypothesized that it would show the same stimulus-goal associations as the stop-signal task. We noted an important difference: Whereas the stop signal is usually presented with all possible go stimuli in the stop-signal task, so the stop goal is relevant to all go-task categories, the stop goal is only relevant to one category in the go/no-go task. Thus, there is consistent mapping of stimuli onto goals in the go/no-go task, so associations between stimulus categories and stopping should become very strong. We demonstrated this in several experiments (Verbruggen & Logan, 2008b). We also showed that strong associations can develop in the stop-signal task if there is consistent mapping of go-task stimuli and stop goals (Verbruggen & Logan, 2008b).

Our research also addressed the methods by which post-stop-signal slowing should be assessed, noting that slow drifts in response time over the course of the experiment can bias estimates of post-stop-signal slowing if researchers use the wrong baseline (Nelson et al., 2010). Typically, researchers use mean go response time on trials without stop signals as the baseline and calculate post-stop-signal slowing by subtracting this baseline from go response time following successful and unsuccessful inhibition. We noted that subjects often fail to inhibit when response time is fast and often succeed in inhibiting when response time is slow. If fluctuations in speed are not taken into account, the data will suggest that there is greater post-stop-signal slowing following successful inhibition than following failed inhibition. We suggested that the go trial before the stop signal trial was a more appropriate baseline. When post-stop-signal slowing is assessed with this baseline, subtracting go response time from the trial before the stop signal from go response time from the trial after the stop signal, there is an equal amount of slowing following failed and successful inhibition (Bissett & Logan, 2011; Nelson et al., 2010).

Our research also addressed the neural basis of post-stop-signal slowing, measuring the activity of movement related neurons in the frontal eye fields and superior colliculus of macaque monkeys performing the stop-signal task (Pouget et al., 2011). Activity in these movement-related neurons normally rises to a threshold on trials without a stop signal, and an eye movement is released when the activity reaches a threshold. On stop signal trials, the activity is attenuated. If the attenuation is sufficient to prevent the activity from reaching threshold, the response is inhibited. If it is not sufficient to prevent the activity from reaching threshold, response inhibition fails. We examined activity in these cells on trials following stop signals to determine which parameters of their activity accounted for the post-stop-signal slowing that was apparent in the monkeys' response times. Typically, there is stable baseline activity before the trial and in the initial part of the trial. Then activation begins to increase until it reaches threshold. We measured baseline activity, the point at which the rise began (the onset of activation), the rate at which activation rose, and the threshold at which a response was inhibited. We found differences in the onset of activation but no differences in baseline, rate of rise to a threshold, or threshold. This result was surprising because stochastic accumulator models of response time typically assume that control adjustments are accomplished by changing response caution, which entails changing baseline or threshold. We also fit the linear ballistic accumulator

model to the monkeys' response time distributions and found that changes in non-decision time accounted for post-stop-signal slowing, consistent with our neural measures. Thus, we found strong convergence between neural measures and computational models.

We have also been exploring the neural basis for visual search, using recorded neural activity as a proxy for drift rate in a stochastic accumulator model and trying to fit response time distributions for correct and error responses and neural activity at the same time (Purcell et al., 2010, 2011; Schall et al., 2011). The data we addressed came from macaque monkeys performing a difficult visual search task in which the number of items in the display varied from trial to trial. Monkeys had to maintain fixation until they found the target and then move their eyes directly to the target. Response time and error rate increased with the number of items in the display. Firing rates were recorded from two kinds of neurons in frontal eye fields: visual neurons that responded to the salience of objects in the visual field, responding to targets and distractors without producing eye movements, and movement neurons, which we also studied in the stop-signal task, that produced eye movements when their firing rates reached a threshold. We used activity recorded from visual cells as the input to response accumulators, having one accumulator for each object in the display, and integrating visual-cell activity in the accumulators until it reached a threshold. We varied the amount and nature of competition between accumulators, comparing feedforward and lateral inhibition and evaluating the role of leakage. We found several models that accounted for response time distributions for correct and error responses rather well, but many of them predicted the wrong baseline activity for movement cells. To capture the dynamics of movement cell activity, we proposed a gate between the visual cells and the movement cells that would block activity from the visual cells until it was strong enough to overcome the gate, whereupon it began to activate the movement cells. This gated accumulator model accounted for behavioral and neural data very well.

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